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CONCERNING KERNIG'S SIGN IN MENINGITIS.[1]

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IN 1884 Kernig,[2] of Russia, described a phenomenon which he thought was found only in affections of the pia mater, and which was never lacking in inflammations of that membrane. If a patient with meningitis be made to sit up, as on the edge of the bed, the thigh therefore being at a right angle with the body, it is found extremely difficult to extend the leg because of the presence of a marked flexor contracture. To quote Kernig: "The phenomenon is so striking, the difference between nothing and something ('zwischen null und etwas'), between the complete absence of the contracture while the patient is lying down, and its presence when the patient sits up, is so plainly perceptible that it is well worth while to pay special attention to this symptom and to examine for it in every case."

Kernig saw fifteen cases of meningitis, in all of which he found the symptom. In eight cases there was confirmation of the diagnosis by autopsy. Of the fifteen cases thirteen were epidemic cerebro-spinal meningitis, one tuberculous, one suppurative. Kernig failed to find the sign in many diseases other than meningeal affections. In six cases, however, where there was not acute meningitis, the sign was more or less marked; yet in all these he found some pial trouble—œdema, intermeningeal hemorrhage, circumscribed meningitis, chronic meningitis, general carcinosis. Kernig believed the sign appeared in meningitis as early as the rigidity of the neck, and that it is late to disappear.

Kernig's sign seems to have attracted comparatively little attention for fourteen years. Most of the standard text-books on nervous diseases make no reference whatever to it. Some authors describe the phenomenon, but disclaim any individual responsibility for the correctness of the statement by bracketing Kernig's name after the statement (*e. g.*, Oppenheim). Still others state briefly that it is rarely found. "A peculiar, but as it appears extremely uncommon, symptom is the so-called flexor contracture of Kernig." (Hirt, p. 14.) Until Netter, in 1898, revived interest in the subject there are, so far as I can discover, but four writers who recorded the results of their clinical observations on this phenomenon: Bull (1885), Henoch (1886), Friis (1887), and Blümm (1889).

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[1] Read at the fourteenth annual meeting of the Association of American Physicians, at Washington, May 2, 1899.

[2] Kernig: Berliner klinische Wochenschrift, December 29, 1884.

Bull[1] wrote to show that the sign was not pathognomonic of meningitis or even of pial trouble, but merely indicated an increase in intracranial pressure. He found the flexor contracture in three cases, one of tuberculous meningitis with no autopsy, one of solitary tubercle of the cerebellum, but "in the pia over it a few miliary tubercles," with tuberculosis in the lung, lymph-glands, kidney, and intestine; one of otitis media with caries of petrous portion, thrombosis of transverse sinus, and common jugular. In the basal pia, especially over the frontal lobe, were ecchymoses, and there were a few teaspoonfuls of serum in the posterior fossa. These cases, it seems to me, go rather to prove Kernig's statement, that his sign is found with affections of the pia mater, than Bull's, that it is not indicative of meningitis or even of involvement of the pia; for, on Bull's own showing, one of his three cases was tuberculous meningitis, another had miliary tubercles in the meninges, the other certainly some local meningitis, with numerous ecchymoses and an unusual amount of serum.

The most extended observations are those of Friis,[2] of Copenhagen. During an epidemic of cerebro-spinal meningitis the examination for Kernig's phenomenon was made in sixty out of 110 cases. In fifty-three of the sixty the sign was present—*i. e.*, in 88 per cent. In two of the remaining seven it was doubtful, in two the examination was unsatisfactory, in three the sign was absent. Friis was unable to state how early it appeared or how late it remained. He found no relation between it and stiffness of the neck, hyperæsthesia, or pain, though he was inclined to think it was more pronounced where lumbar pain was most complained of. Friis has no explanation to offer as to the cause of the phenomenon, but does not agree with Bull in regarding it as due solely to intracranial pressure, as he has seen it absent in a case of hydrocephalus.

[1] Eduard Bull: Berliner klinische Wochenschrift, 1885, No. 47, p. 772. Ueber die Kernigsche Flexionscontractur der Kniegelenke bei Gehirnkrankheiten.

[2] Albert Friis: Om den i Kjobenhavn i Aaret, 1886 Herskende Epidemi af Meningitis Cerebrospinalis. Thesis, 1887, p. 154.

In an epidemic in Copenhagen in 1891 Friis[1] found the sign in twenty-one of twenty-six cases (80 per cent.).

Henoch,[2] in 1886, reported on the Kernig sign. His later views can be gathered from his reference to it in his *Kinderkrankheiten*, seventh edition, p. 320. He has found the Kernig contracture present in many cases of meningitis, but does not regard it as pathognomonic, as he has in at least one case with autopsy found no contracture; and he thinks it may be present without meningitis.

Blümm[3] found Kernig's sign in seven out of nine cases of meningitis.

Last year, 1898, Netter,[4] in examining twenty-five cases of meningitis, found Kernig's sign present in twenty-three (92 per cent.), some cases abortive, others advanced, two convalescent. Eight cases were tuberculous, three mixed tuberculous and epidemic, twelve epidemic. One of the exceptional cases was a child seen but once, the other a child living at the time of the communication with evidences of advanced tuberculous meningitis. Netter believes its presence permits one to assert quite positively the existence of a meningitis. He regards it as of especial value in the diagnosis of abortive types of epidemic meningitis. From its persistence into convalescence (Kernig, Henoch, Netter) it may be of service in enabling one to make a retrospective diagnosis.

A still more extended experience with forty-seven cases is reported by Netter.[5] He found the test positive in about 90 per cent. of cases of meningitis.

My own observations have been made upon nineteen cases of meningitis, with autopsy in eight cases. All doubtful cases have been excluded. Nine were epidemic cerebro-spinal meningitis, seven were tuberculous, two were pneumococcal, one was syphilitic with an acute process superadded. These cases, for convenience of reference, have been arranged in tabular form. I take this opportunity of thanking the physicians who gave me the opportunity of examining some of their patients, and in particular Drs. Billings and Preble, who courteously extended to me the freedom of their wards at the Cook County Hospital.

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[1] A. Friis: Ugeskrift for Laeger, 1892, pp. 407 and 431, cited by Netter.

It is interesting to note that in studying these cases Friis found and described an intracellular diplococcus evidently the intracellularis. His figure on page 27 of his thesis is an excellent representation of the specific organism. He does not, however, declare it to be the specific germ, though questions its identity with Fränkel's pneumococcus.

[2] Henoch. Charité Annalen, 1886, p. 575.

[3] Blümm: Münch. med. Woch., June 25, 1889.

[4] Netter: La Semaine Médicale, 1888, p. 281. Clinical lecture delivered at Hôpital Trousseau.

[5] Bulletin et Mémoires de la Société Médicale des Hôpitaux de Paris, 22 Juli, 1898. Abstract in Deut. Med. Woch., Jan. 26, 1899.

TABLE I.—SHOWING CASES OF MENINGITIS EXAMINED FOR KERNIG'S SIGN.

No.	Date.	Name, age, sex.	Where seen.	Day of disease.	Clinical symptoms.	Kernig's phenomenon.	Result.	Autopsy.	Remarks.
1	Sept. 30, 1898	.... adult, F.	C. C. Hospital, Dr. Preble.	?	Fever, delirium, rigid neck, swollen joints, paralysis of some of ocular muscles.	Marked.	Died.	Pneumococcus septicæmia, with suppurative arthritis, pericarditis and meningitis	
2	Oct. 7, 1898	? adult, color'd, M.	C. C. Hospital.	12th	Old cough, slightly dull at one apex, chest elsewhere hyper-resonant, emaciation, cyanosis, rapid respiration, fever, delirium; T.B. in sputum. Diagnosis; miliary tuberculosis. On Oct. 5th, no rigidity of neck and no Kernig; on Oct. 7th, neck rigid and retracted, and Kernig pronounced.	Marked; can lift patient from bed without the knees yielding in extension.	Died.	No autopsy.	Of interest to note absence of Kernig at first, but appearance later as evidence of meningeal involvement became marked.
3	Oct. 16, 1898	R. adult, M.	Drs. McNeal and Heywood.	8th	Headache, vomiting, temp. 99-102°, delirium, insomnia, involuntaries; no spleen; neck rigid, facial paresis; no evidence of syphilis, no benefit from iodides.	Present.	Died.	No autopsy.	Meningitis, probably tuberculous.
4	Oct. 30, 1898	.... Adult, color'd, M.	C. C. Hospital.	?	Fever, semicoma, rigid neck, right ptosis, etc. Diagnosis: Tuberculous meningitis.	Present.	Died.	Autopsy: tuberculous meningitis.	
5	Nov. 15, 1898	Willie, 10 yrs. M.	C. C. Hospital.	9th	Headache, delirium, later coma; strabismus, rigidity and retraction of neck, unequal pupils, neuro-retinitis, temp. 99-101°, herpes labialis.	Present.	Died.	Autopsy: meningitis.	Nature of meningitis not determined bacteriologically; probably epidemic.
6	Dec. 2, 1898	.... adult, M.	C. C. Hospital.	?	Alcoholic and syphilitic; headache, thick speech, rigid neck and jaw, involuntaries, emaciation, cyanosis, rapid respiration. A micrococcus in smears from lumbar puncture.	Present.	Died.	Autopsy: syphilitic meningitis with acute process superadded.	

7	Dec. 6, 1898	M. 48 yrs. M.	C. C. Hospital.	?	Brought in unconscious; temp. 101°, hyperæsthesia, rigidity of neck; diplococcus by lumbar puncture; mind clearer after lumbar puncture.	Marked.	Died.	Autopsy: cerebro-spinal meningitis.	
8	Dec. 17, 1898	W. 4 yrs. F.	Dr. Pigall.	14th	Prodromal irritability, headache; five days ago convulsion; later rigid and retracted neck, and coma; pupils unequal; Cheyne-Stokes respiration, emaciation, patellar reflexes slightly exaggerated.	Not present, or very slight	Died within a few hours after examination.	No autopsy.	Diagnosis: tuberculous meningitis.
9	Jan. 11, 1899	.... 25 yrs. F.	Dr. Urheim.	7th	Empyema one year before; operation; two later operations, but sinus still remaining; for several days headache, bewilderment, delirium, temp. 100°; no rigid neck or paralysis at time of examination.	Present.	Died two days later.	No autopsy.	Probable tuberculous meningitis secondary to primary pleural tuberculosis.
10	Jan. 16, 1899	S. 8 yrs. M.	Dr. McCullough	8th	Influenza, pneumonia, suppurative otitis; for about four days stupor; temp. 100 to 104°; constipation, retention of urine; neck retracted and stiff; lung still consolidated; no paralysis, but great laxity of all muscles; only by severe pinch or firm supra-orbital pressure can even a slight turning of the hand be produced; no patellar reflexes.	Not present in right leg, in left very slight.	Died 20 hours after examination.	No autopsy.	Pneumonia, metapneumonic otitis and meningitis.
11	Feb. 20, 1899	B. B. 27 yrs. F.	C. C. Hospital.	Several	Two years ago nursed husband with consumption; one year ago pleurisy. Before admission severe headache and convulsions, delirium, strabismus, retinitis, retracted neck, "ganglion" on hand; lumbar puncture negative; cyanosis, rapid respiration, tender abdomen, temp. 99-102°.	Present.	Died.	Autopsy: old tuberculous pleurisy, solitary tubercles in brain, with general miliary tuberculosis; some small tubercles in meninges.	
12	Feb. 22, 1899	.... adult, M.	C. C. Hospital, Dr. Billings.	Several	Sore-throat, headache, delirium, herpes, petechiæ, retracted neck, diplococcus intracellularis from cerebro-spinal fluid; coma on eighth day.	Marked.	Died.	Autopsy: epidemic cerebro-spinal meningitis.	
13	Feb. 26, 1899	S. 17 yrs. F.	Dr. T. E. Roberts	17th	Tuberculous family history; prodromal irritability of temper; headache, becoming extremely severe and constant, temp. for 17 days 99-103°; delirium, retraction of neck, variable pulse, ptosis, Cheyne-Stokes, coma, rapid pulse; no spleen, spots or Widal.	Present in lt. leg; in rt. leg, by constant, gentle force, the leg straightens out, giving way with a sort of snap.	Died, 56 hours after examination.	No autopsy.	Diagnosis: tuberculous meningitis.

No.	Date.	Name, age, sex.	Where seen.	Day of disease.	Clinical symptoms.	Kernig's phenomenon.	Result.	Autopsy.	Remarks.
14	Mar. 22, 1899	A. L. 8 yrs. M.	Presb. Hosp., Dr. Cotton.	20th	Headache, vomiting, delirium, rigid neck, coma, variable pulse; temp. 99-103°+; leucocytosis (22,000); lumbar puncture negative; great emaciation.	Present continuously from Mar. 22 to May 2.	Recov., with deafness.	....	Diagnosis: epidemic cerebro-spinal meningitis
15	Mar. 24, 1899	V. 25 yrs. F.	C. C. Hospital, Dr. Roberts.	7th	Headache, tender and rigid neck, muscular twitchings, tâche cerebral, delirium; temp. 100-104°; lungs, heart, etc., negative.	Marked.	Died.	No autopsy.	Diagnosis: meningitis, probably epidemic.
16	Mar. 24, 1899	W. B. adult. M.	C. C. Hospital, Dr. Preble.	?	Coma, restless, rigid neck, slight ptosis; temp. 103–104°, pulse 60–88, albumen; an abundant diplococcus by lumbar puncture.	Marked; Kernig also present just before death, though not as marked as day before.	Died.	No autopsy.	Epidemic meningitis.
17	Mar. 25, 1899	C. R. 41 yrs. M.	C. C. Hospital, Dr. Billings.	?	Headache, slight rigidity of neck, stupid even to falling out of bed, involuntaries; temp. 98-100°; diplococcus by lumbar puncture.	Present.	Recov.	....	Probable epidemic meningitis.
18	Mar. 26, 1899	Geo. S. color'd, 30 yrs. M.	Dr. F. B. Earle.	29th	Onset with headache, vomiting; delirium, retracted and rigid neck, pulse rarely over 100, temp. 97-104°, involuntaries, resp 24-34; no cyanosis, leucocytosis; no plasmodia, no Widal.	Present.	Recov.	....	Epidemic cerebro-spinal meningitis; on May 1st, there was no Kernig present; patient able to walk.
19	April 2, 1899	N. 2½ yrs. F.	Dr. Pigall.	3d	At first feverish, wanting to lie down; mother thought "billous" and gave oil; next day convulsions and fever, stupor, "gritting of teeth," jerkings, coma, eyes half open, left eye reddened and with muco-pus on globe, neck rigid when attempt is made to move it, and whole body then quivers; temp. 102°+; pulse 160, respiration irregular, lungs negative.	Present.	Died, in 12 hrs.	No autopsy.	Meningitis, probably epidemic.

From the table it will be seen that Kernig's sign was present in seventeen out of nineteen cases of meningitis, or in 89.4 per cent. In the two cases in which it was absent, both children, the single examination was made a short time before death, and it was noticed at the time that there was a general marked laxity of all the muscles, in the one case there being but the faintest movement of an arm in response to firm and long-continued supraorbital pressure. This recalls the fact that even the retraction and rigidity of the neck muscles may be variable, and just before death may entirely disappear. "In patients deeply comatose and shortly before death I have frequently seen the stiffness of the neck disappear."[1] Kernig's sign may have been present earlier. Friis states that it may vary in degree from day to day. In most of the cases examined the other signs were so unequivocal as to make the examination for the Kernig phenomenon wholly unnecessary except as a confirmatory finding. In some of the cases, however, its presence was of distinct value from the paucity of the common evidences of meningitis—*e. g.*, in cases three and nine. And in case two—one of miliary tuberculosis—it was of special interest to note the absence at first of Kernig's phenomenon and its later appearance at the same time with the other evidences of meningeal involvement—retraction and rigidity of neck, severe headache, paralysis of ocular muscles.

Netter,[2] in a footnote, cites an extremely instructive case showing the value of the Kernig phenomenon. In a typhoid with typical symptoms, including the agglutination test, Kernig's sign was marked. But from the absence of other pronounced meningeal symptoms meningitis was excluded. Death occurred in the third week from perforation. The autopsy revealed typhoid, and also a cerebro-spinal meningitis due to the staphylococcus pyogenes and the bacillus typhosus.

Many patients with diseases other than meningitis were examined as to the existence of Kernig's phenomenon, there being selected as many cases as possible in which there were cerebral symptoms. Many individuals of all ages who were in apparently perfect health were examined. In twenty-five healthy individuals selected at random no Kernig sign was present. In 100 cases of disease other than meningitis Kernig's sign was absent in ninety-eight and present in two. The result is shown in the following table:

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[1] Eichhorst. 4th edition, iv. 473.

[2] Netter. La Semaine Méd., 1898, p. 282.

TABLE II.—SHOWING CASES OTHER THAN MENINGITIS EXAMINED FOR KERNIG'S SIGN.

No. of cases examined.	*Diagnosis.*	*Kernig's phenomenon.*
25	Healthy individuals from infancy to old age, both thin and fleshy	Absent.
5	Pneumonia	"
3	Convalescent pneumonia	"
2	Pneumonia with delirium tremens	"
5	Delirium tremens	"
1	Tetanus; fourteen days after injury, trismus, convulsions, fever, mild delirium	"
2	Uræmia	"
3	Malaria	"
1	Convalescent malaria	"
3	Typhoid fever	"
5	Cerebral hemorrhage	"
1	Cerebral thrombosis	"
3	Cerebral syphilis	"
1	Tumor of brain	"
1	Subdural hemorrhage (autopsy)	Present.
1	General paralysis of insane	Absent.
1	Multiple sclerosis with spasticity	"
1	Ataxic paraplegia	"
2	Convalescent multiple neuritis	"
1	Tabes and hysteria	"
2	Hysteria	"
1	Incipient Graves's disease	"
2	Lead poisoning	"
1	Opium poisoning	"
1	Influenza with cerebral symptoms	"
1	Pernicious anæmia	"
2	Chlorosis	"
1	Enteroptosis	"
4	Acute rheumatism	"
4	Chronic rheumatism	"
1	Gonorrhœal rheumatism (vide text)	Present.
10	Valvular disease of heart	Absent.
1	Fibrous myocarditis	"
1	Acute endocarditis	"
5	Carcinoma of stomach	"
2	Ulcer of stomach	"
3	Asthma and bronchitis	"
1	Mastitis	"
1	Chronic nephritis	"
1	Amœbic dysentery	"
6	Pulmonary tuberculosis	"
1	Pelvic inflammation	"
1	Facial erysipelas with delirium, vomiting, high fever, headache, etc.	"
1	Loose body in knee-joint	"
1	Chronic gastritis	"

In a case of Dr. Billings, Kernig's sign was present where post-mortem a subdural hemorrhage was found with no inflammatory changes. The patient was a laborer, aged fifty-two years. Headache and forgetfulness for a few days. Found unconscious, having apparently fallen out of bed. Rigid and retracted neck, equal pupils, right facial paresis, right arm flexed and rigid. Involuntaries. Temperature on admission normal, but in twenty-four hours up to 104°, with pulse 130. After lumbar puncture temperature 98°, pulse 56. Later temperature but slightly elevated. Pulse variable. Mental condition varied from coma to quite bright intelligence. When ratonal no rigidity and no Kernig sign. When comatose Kernig's sign present. Died in coma on fourteenth day. Autopsy revealed subdural hemorrhage, left side.

It was present in the unaffected leg in a woman who had been lying in bed for four weeks with a gonorrhœal arthritis of one knee. During this time she had lain with both knees flexed, as this was the most comfortable position. This local condition may explain the difficulty experienced in making extension in the leg so long accustomed to flexion.

The absence of the sign was of diagnostic value in the case of facial erysipelas. The patient, an adult, went to work in the morning, feeling nearly as well as usual, and returned at noon complaining bitterly of headache and nausea. "He staggered into the house like a drunken man." By 3 P.M. he was delirious, and his physician found a temperature of 104°, with, he thought, some stiffness of the neck, and diagnosed meningitis. When I saw him the next day there was still complaint of severe pain in the head; he had not slept the night before; was slightly delirious; temperature 103°. Other findings in the lungs, heart, abdomen, muscles, negative, save that breathing through the nose was difficult, as the nose seemed "stuffed up" as from a cold. There was no Kernig sign, though the patient, a fleshy man, and his wife both said they knew he would be unable to extend the leg as he sat on the edge of the bed, as he had tried it many times in his efforts to put on his shoes and stockings. In one leg extension was made with some little difficulty, but still with sufficient ease to exclude Kernig's phenomenon—a strong argument, it seemed to me, against meningitis. The next morning the nose and adjacent skin of the face showed typical erysipelas, and the case was clear, though the prevalence of sporadic cases of epidemic meningitis made one at first seriously consider the possible existence of this affection.

From the study of cases made by Kernig, Bull, Henoch, Friis, Blümm, and Netter, with the observations just recorded, it seems justifiable to regard Kernig's phenomenon as present in the majority of cases of meningitis—say 80 to 90 per cent.—and as only exceptionally present in other affections. Other and more definite conclusions concerning it seem unwarranted at present. That it may occasionally be absent in meningitis and occasionally present in diseases other than meningitis should not cause it to be wholly discarded as of no value. The truly pathognomonic symptoms are few. Rose-spots in typhoid, herpes in malaria and pneumonia, absence of free hydrochloric acid in carcinoma of the stomach, murmurs in endocarditis, choked disk in cerebral tumor, and scores of other symptoms are not pathognomonic, but rightly deserve attention, when found or when absent, as helping to make up or modify the symptom-complex of a disease, the method of diagnosis upon which as yet clinicians are compelled largely to rely.

As to the technique of eliciting this sign, it is exceedingly simple. The patient may be made to sit up on the edge of the bed with the legs hanging down, thus bringing the thigh at right angles to the body, when extension of the leg can be attempted; or the thigh can be brought at right angles to the body as the patient lies in bed on the side, or preferably on the back, and the leg then extended. The only requirement seems to be that the thigh should be placed at a right angle with the body before the attempt is made to extend the leg. Not infrequently in delirious patients—*e. g.*, in delirium tremens—a little, patient, gentle force must be used before the jerking and tense muscles yield. The same is true of some spastic conditions. But where Kernig's contracture is present patience and gentleness do not cause the muscles to yield, and the back of the patient can generally be lifted from the bed without the knees giving way. This procedure is often accompanied by evident pain to the patient. In some cases the leg and thigh will make an obtuse angle of no more than 110° or 120°. As Kernig says, the test in many cases is most striking, particularly in those patients where thigh and leg in the recumbent position are extended and apparently lifeless, but in the test position reveal a most intensely rigid contracture, often with evidence of acute pain. In testing for Kernig's sign there must be excluded those cases in which local causes could interfere with the proper extension of the leg, such as rheumatic or other form of arthritis of the hip or knee, myositis, old contractures from nervous diseases, and sciatica. The phenomenon can be well imitated by the latter condition.

No thoroughly satisfactory explanation of this phenomenon has been offered. Bull's notion that it is merely an evidence of increased intracranial pressure is disproven by the many failures to find the sign in hydrocephalus, in tumors, and in hemorrhage. His notion is, further, that it is but an exaggeration of a normal condition. Even in health it is often somewhat difficult to secure easy and complete extension of the leg with the thigh flexed at right angles with the body, owing to the tenseness of the muscles at the back of the thigh. Henoch[1] aptly cites the statement of Landois that the flexors of the leg springing from the tuber ischii are too short to permit of free extension of the leg when the thigh is flexed on the body at an acute angle, and he further calls attention to the fact, noted by Henke,[2] that owing to the shortness of their muscular fibres and the abundance of tendinous fibrous material the semi-tendinosus, semi-membranosus, and biceps femoris are unable to stretch far enough to permit of complete extension of the leg when the thigh is sharply flexed on the body. It is easy to assume, therefore, that in meningitis there is such an increase in tone as to exaggerate this natural difficulty of extension into a contracture of the flexors of the leg. But just how this is brought about is a matter of speculation.

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[1] Loc. cit.

[2] Henke. Hand Atlas, Berlin, 1888, i. 175.

ON ASTASIA-ABASIA, WITH A CASE.

By J. C. Wilson, M.D.,
of Philadelphia.

It is a remarkable fact that the syndrome astasia-abasia has attracted so little attention in America. The earliest account of the condition, according to Knapp,* was given by Jaccoud in 1864. Weir Mitchell, in 1881, reported a case under the heading "Hysterical Motor Ataxia." Knapp's very complete paper was published in 1891. Up to that time he had been able to collect only forty-nine cases, in addition to his own, which presented "some of the symptoms of abasia, but which is not wholly typical." Of these cases thirty had been reported by French observers, and only four had been studied in this country. These were: 1. The case of Weir Mitchell, already referred to (1881). 2. That of Hughes (1890). 3. That of Hammond (1891). 4. That of Knapp (1898). Dr. Kalteyer has been good enough to look up for me the references to this curious and rare nervous disturbance since the date of Knapp's paper. He finds in the *Index Medicus* forty-three separate titles. Of these communications twenty-two are by French or Belgian and five by American physicians. The American papers are those of: 1. M. Prince (1892); 2. Bremer (1893); 3. F. R. Smith (1894); 4. Eshner (1896); 5. Diller (1896). To the nine cases hitherto reported by American observers I desire to add one recently observed in private practice.

A woman, aged twenty-four years, of good position and in comfortable circumstances, is the subject of the following note: She comes of distinguished stock, but on the paternal side there is an uncle epileptic and an aunt addicted to morphine; on the maternal, two uncles dipsomaniacs. Her father was very successful in business, had grandiose ideas, and lost a large fortune. He is now greatly absorbed in tracing geneologies. Her mother died of diphtheria. She had a sister who died of diphtheria at an early age, and one older than she who is in excellent health, but excitable. The patient had the diseases of childhood severely, but grew up in good health. She has for some years, since the death of her mother, kept house for her father, who is much preoccupied, but she has had her own friends, and is very intelligent and cheerful. Lately there was an unsatisfactory love affair. Up to this time her health had been excellent. Early in January she began to suffer from bath pruritus. January 16th, being at the close of menstruation, she was roused from sleep at midnight by a ring at the bell. She hurried into her wrapper and slippers and opened the door to receive an indifferent telegram from a member of the family in another city. The next day, on attempting to rise, she found it impossible to stand or walk. With the assistance of her maid she could cross the room, but unsupported she sank to the floor. She described her condition in the following terms: "My left leg feels as though it were made of cotton"—an expression already recorded—and "I feel like a combination of a child learning to walk and a drunken woman." Her people were greatly alarmed, but she treated the matter lightly. In the course of a day or two the following clinical data were collected:

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* Journal of Nervous and Mental Diseases, New York, November, 1891, xvii. No. 11.

The patient stated, referring to her matrimonial engagement which had been broken off, that for some months prior to the attack she had been worried. She was slightly paler than usual, but refused to have a blood examination. She had menstruated regularly and without pain. Appetite fair; digestion satisfactory; bowels slightly constipated; up to this time vomiting had not occurred. There were no signs of visceral disease. The urine is normal. Lying in bed or seated upon a chair, the lower extremities presented nothing abnormal; motor power, freedom and precision of movement, and the muscular senses were unimpaired; knee-jerks decidedly increased; ankle clonus not present; the plantar reflex intact; electrical reactions normal. There was no rigidity, ataxia, or paralysis. Sensation to touch, temperature, and pain appeared to be unimpaired. The patient complains, however, of subjective sensations of numbness and pricking throughout the left lower extremity, including the foot, leg, and thigh. Similar sensations are referred to the foot and outer surface of the right leg, but they are not felt above the knee. Upon attempting to stand her legs bend under her, and unless supported she sinks to the ground. The irregular movements of the limbs are repeated much in the same manner at each effort to stand. They correspond closely to the description of Blocq.* There are abrupt movements of flexions of the knees, followed by rapid extension; these contortions necessitate compensatory movements of the upper extremities and the head in order to maintain the equilibrium. Upon attempting to walk with some support these difficulties are increased; the body is thrown forward, the thighs and knees are abruptly flexed, and then extended. These movements are wholly irregular. With some support she is able to move a short distance, but presently her legs bend under her and she sinks to the floor. She volunteers the statement that in order to obtain an article she desired she was able to cross the room upon her hands and knees.

The patient was kept in bed and not allowed to see members of her family or friends. Valerianate of zinc, in one-grain dose four times a day, was administered in gelatin-coated pills. She objected strongly to the odor of the pills, but nevertheless took them as prescribed. Once a day she had massage of the legs and feet for forty minutes; once a day faradism with the electrical brush for twenty minutes, the current falling just short of pain. At the end of two weeks she was allowed to get out of bed. The difficulty in standing and walking was much less. She was now given lessons in standing and walking. Her nurse was experienced and intelligent, and entered fully into the plan of the treatment. Improvement was rapid, and in ten days the patient was able to go about her room, and, assisting herself by the baluster, to go down stairs for a drive. In a few days the motor disturbance and the paræsthesia had wholly disappeared. About this time distinct hysterical phenomena—vomiting, intermittent tremor of the hands, vertigo, sensations of throbbing in the abdomen, emotional manifestations, weeping, and excitement without apparent cause—manifested themselves. There was no spinal tenderness; no tenderness over the so-called ovarian regions, and hystero-genetic areas were not found.

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* Paul Blocq. Revue Generale de Clinique et de Thérapeutique, March 14, 1889, vol. iii. No. 11.

She was again isolated and placed under a systematic regimen. In the course of a month great improvement occurred, and it was thought well to send her to visit to some friends at a distance who knew nothing of her sickness or its cause.

This case corresponds to the description of the condition by Blocq, who, in 1888, collated the instances, numbering at that time eleven, and gave to the affection the name astasia-abasia, now generally received. This observer defined the condition as " a morbid state in which the impossibility of standing erect and walking normally is in contrast with the integrity of the sensibility, of the muscular strength, and of the co-ordination of the other movements of the lower extremities." (Knapp). With those who are familiar with Blocq's papers and that of Knapp it is quite unnecessary to discuss the theories of the causation of this condition. Church's statement that it is due to a systematized amnesia embodies that theory which is generally accepted. That astasia-abasia is not a nosological entity, but a syndrome, must be conceded. In the foregoing case it was a symptom of hysteria. That it is usually such a symptom, most of those who have seen cases believe. Osler speaks of it as " a manifestation of a neurosis allied to hysteria." Many of the cases have occurred in connection with organic disease. This fact does not militate against the hysterical origin of the condition, since cases of organic disease of the nervous system are often associated with hysteria.

In closing I would call attention to the expressions used by my patient in describing her condition: " That her left leg felt as though it was made of cotton," and that she " felt like . . . a baby learning to walk." It is very remarkable that this girl, who has no knowledge of medicine or medical books, should have used the very phrases so often encountered in the writings upon the subject and at once descriptive o the condition and suggestive in view of the generally accepted theory of its causation.

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